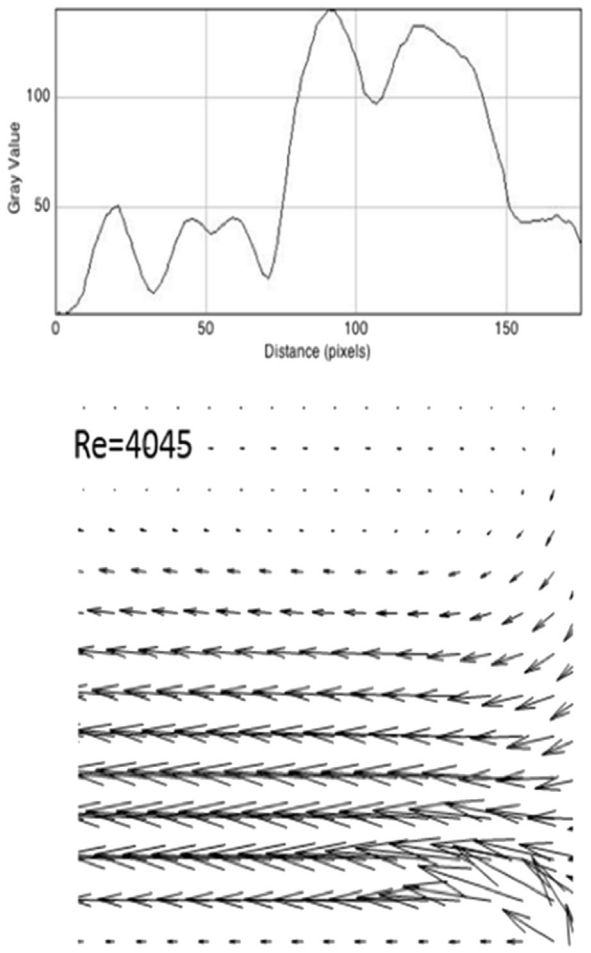

Fig 1. Low intrinsic renal resistance.

underlying cause of both renovascular hypertension and the development of renal artery stenosis may be increased resistance in distal renal arteries and arterioles.

Methods: We examined the velocity spectrum of 20 widely patent renal arteries. The contralateral renal artery was normal in 10 of these and stenotic or stented in 10. We compared the spectral broadening in these arteries by analyzing their mean to peak systolic flow velocities to see if patients prone to develop renal artery stenosis had more of their blood flow at lower velocities, which would be expected if these arteries had lower shear stress and eddy currents. We examined whether distal renal artery and arteriole resistance, which would result in decreased glomerular pressure and renovascular hypertension, would also account for blood flow effects inducing the development of proximal renal artery stenosis in a fluid dynamic model.

Results: Normal renal arteries when the contralateral artery was also normal had a mean to peak systolic velocity ratio of 0.61 vs 0.40 in normal renal arteries when the contralateral renal artery had stenosis or had been previously stented ($P < .001$ by *t*-test). Similar spectral broadening, areas of low shear stress, and eddy currents were induced in the fluid dynamic model as the coefficient of resistance was varied to simulate increasing distal arterial resistance. Fig 1 shows a narrow spectrum with streamlined flow with low distal renal arterial resistance. Fig 2 shows spectral broadening with altered shear stress and flow as distal renal arterial resistance is increased (right).

Conclusions: There is strong evidence that increased distal renal arterial and arteriolar resistance may a primary cause not only of renovascular hypertension but may also lead to the development of proximal renal artery stenosis. Proximal renal artery stenosis may often be an effect rather than a cause of renovascular hypertension explaining why its treatment may be ineffective.

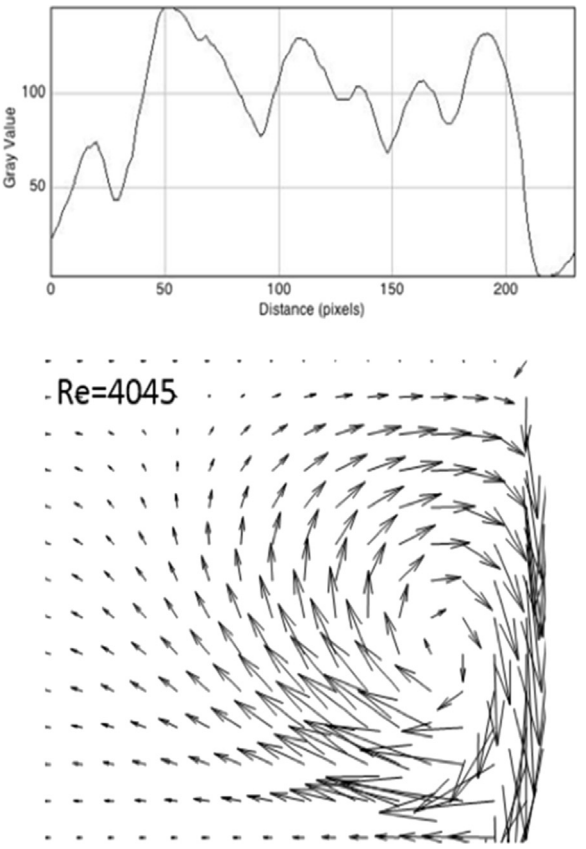

Fig 2. High intrinsic renal resistance.

Author Disclosures: A. P. Sawchuk: None; **H. Yu:** None; **M. C. Dalsing:** None.

Epidemiology of Thoracoabdominal Aortic Aneurysm Repairs in the United States from 1998 to 2011

Sapan S. Desai, MD,[1] Anahita Dua, MD,[2] Mark K. Eskandari, MD,[3] Cheong J. Lee, MD[2]. [1]Duke University Medical Center, Durham, NC; [2]Medical College of Wisconsin, Milwaukee, Wisc; [3]Northwestern University Feinberg School of Medicine, Chicago, Ill

Objectives: The aim of this study was to evaluate and compare national trends in the incidence of unruptured and ruptured thoracoabdominal aortic aneurysms (TAAAs) and their management by thoracic endovascular aneurysm repair (TEVAR) and conventional open aortic repair (OAR).

Methods: A retrospective analysis of the cross-sectional National Inpatient Sample (1998-2011) was used to evaluate patient characteristics and outcomes related to TEVAR and OAR for unruptured (International Classification of Diseases, 9th Revision [ICD-9] 441.7) and ruptured TAAAs (ICD-9 441.6). Data were extrapolated to represent population-level statistics through the use of data from the United States Census Bureau. Comparisons between groups were made using descriptive statistics. Financial data are presented as medians in 2014 US dollars corrected using the Consumer Price Index.

Results: There were 23,081 patients in the National Inpatient Sample affected by TAAAs over the 14-year span of this study. The average patient age was 75 years, 52% were women, and 90% were white. There were 21,727 patients (94%) classified as unruptured and 1354 as ruptured. The overall in-hospital mortality rate for patients with unruptured TAAAs was 7% in 1998 and dropped to 6% by 2011. Median hospital charges for this cohort increased by $11,327 over a decade, from $15,687 in 2001 to $27,014 by 2011 ($P < .001$). For patients with ruptured TAAAs, the overall mortality decreased from 60% in 1998 to 41% by 2011 ($P < .01$). There was an increase in the use of TEVAR to treat aneurysms from 1998 to 2011 compared with OAR, from no cases in 1998 to 177 in 2011 ($P < .001$) regardless of rupture status. TEVAR was associated with an overall mortality of 8% compared with 19% for OAR ($P < .001$). There was an overall decline in

the number of TAAA cases performed nationally and a decline in the rate of ruptured TAAA ($P < .001$).

Conclusions: A decline in the number of TAAA cases was observed over the study period; however, the costs associated with treatment increased significantly. The overall in-hospital mortality is decreasing for TAAAs. A sharp rise in the use of endovascular techniques for the management of TAAA was seen over the study period. The overall mortality of TEVAR was significantly lower compared with OAR.

Author Disclosures: S. S. Desai: None; **A. Dua:** None; **M. K. Eskandari:** None; **C. J. Lee:** None.

Upper Extremity Steal Syndrome Is Associated With Atherosclerotic Burden and Access Configuration

Elizabeth A. Kudlaty, Daniel E. Kendrick, MD, Matthew T. Allemang, MD, Vikram S. Kashyap, MD, Virginia L. Wong, MD. Division of Vascular Surgery & Endovascular Therapy, University Hospitals Case Medical Center, Cleveland, Ohio

Objectives: Clinically significant steal syndrome (SS) occurs in a subset of dialysis patients with arteriovenous (AV) access. Factors associated with SS are poorly understood. Severe symptoms require access revision or sacrifice, potentially jeopardizing access options. Our objective was to review our dialysis access experience to identify factors associated with SS.

Methods: We reviewed all adult patients undergoing their first upper extremity access, AV fistula (AVF) or AV graft (AVG), between January 2008 and July 2011 at a single center. Medical, demographic, and access characteristics were collected from our electronic medical record and a local dialysis center's database. Patients who required correction of SS were compared with the larger access cohort. Statistical analysis included the Fisher exact test and χ^2 for noncontinuous variables and the unpaired *t*-test for continuous variables.

Results: Of 303 patients, 15 required correction for SS (eight of 232 AVF and seven of 71 AVG). Eight were ligated; two were initially banded, then ligated; and five underwent distal revascularization with interval ligation. Coronary artery disease was more prevalent in SS patients (66.7% vs 25%; $P = .001$); the same was found with peripheral arterial disease (40% vs 13.8%; $P = .02$). Further, more patients with SS were on Plavix for cardiovascular reasons (40% vs 9%; $P = .002$). SS only developed with AVF/AVG using brachial artery inflow. No cases of SS arose from radial/ulnar inflow ($P = .03$). All AVGs with SS had a straight configuration; no looped AVG developed steal ($P = .02$). Other patient characteristics such as age, sex, race, hypertension, diabetes mellitus, congestive heart failure, cerebrovascular accident, cause of end-stage renal disease, and other medication history were not different between groups.

Conclusions: Clinically significant SS is associated with disease in coronary and peripheral arterial beds. In addition, the use of brachial artery inflow and straight AVG configuration are associated with SS. Patients with these characteristics need additional vigilance for steal-associated symptoms.

Author Disclosures: E. A. Kudlaty: None; **D. E. Kendrick:** None; **M. T. Allemang:** None; **V. S. Kashyap:** None; **V. L. Wong:** None.

Descending Thoracic Aortic Surgery Is Common After Type A Aortic Dissection Repair: Perspectives From Thomson Reuters MarketScan Database

Ross Milner, MD, Sandra Ham Michael Hur. Surgery, University of Chicago, Chicago, Ill

Objectives: The current standard of surgical repair for type A aortic dissection (TAAD) does not guarantee freedom from subsequent aortic operations; therefore, consideration of simultaneous treatment beyond the ascending aorta is warranted. This study was conducted to report the national rates and the timing of proximal and distal reoperations after operative repair of TAAD based on available patient-level admissions data.

Methods: Admissions data for patients with TAAD were identified and obtained from Thomson Reuters MarketScan Database using International Classification of Diseases, 9th Revision, Clinical Modification codes. From 2003 to 2011, patients aged >18 years with thoracic or thoracoabdominal diagnoses who underwent surgical repair or aortic resection, or both, were identified. From this population, 2041 patients with ≥12 months of follow-up data who were discharged alive after index admissions for TAAD were described. Primary outcome measures were rates of reoperation for any aortic location. Patient demographics, comorbidities at admission, and interval to reoperation were also obtained from the database.

Results: A total of 3463 patients with TAAD who underwent repair were identified. The mortality rate was 7.8% for the initial repair. Of 3018 patients who were available for follow-up, 2041 patients had >1 year of follow-up. The mean age was 58.0 ± 13.2 years, and 70% were male. Eighty-seven patients (4.26%) required 95 reoperations (33.1% of reoperations) for the ascending aorta, and 162 (7.94%) patients required 192 (66.9%) reoperations for the descending thoracic aorta. The mean intervals were 436.7 ± 593.9 days for ascending aortic surgery and 324.0 ± 431.2 days for the descending thoracic aorta after the initial admission for TAAD surgical procedures.

Conclusions: TAAD is a highly morbid and mortal diagnosis. The initial surgery is high-risk, but our data demonstrate that future surgery is common. In fact, the majority of the repeat surgical procedures are for descending thoracic aortic complications. We believe that more aggressive aortic stabilization may be warranted.

Author Disclosures: R. Milner: None; **S. Ham:** None; **M. Hur:** None.

Endovascular Versus Open Repair of Thoracoabdominal Aneurysms: Long-Term Results

James F. McKinsey, MD,[1] Natalia Egorova[2]. [1]Vascular Surgery, Columbia University New York Presbyterian Hospital System, New York, NY; [2]Mount Sinai Medical Center, New York, NY

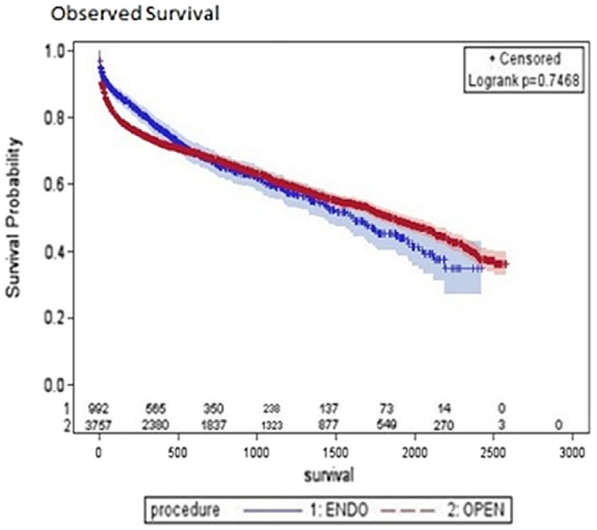

Fig 1. Observed survival.

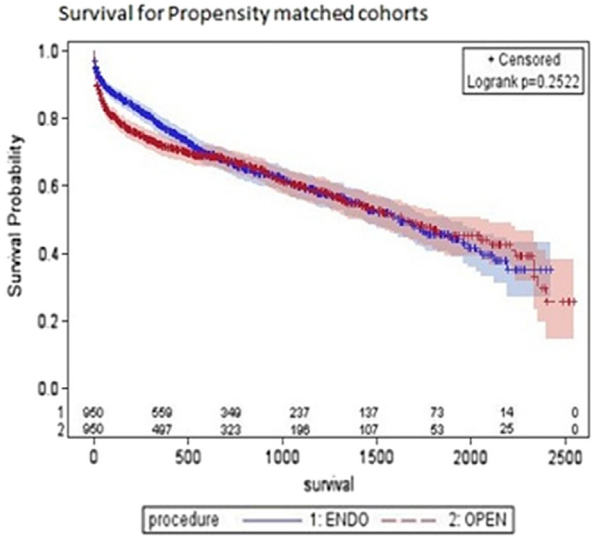

Fig 2. Survival for propensity-matched cohorts.